REMARKS

As can be seen from the pending claims, the present invention relates in part to treatment of acute lung injury resulting from indirect causes which occur systemically. An example of a systemic cause of indirect acute lung injury is septicemia. According to "Taber's Medical Encyclopedia" (attached herewith), septicemia is defined as the spread of an infection from its initial site to the bloodstream" initiating an adverse systemic response.

Folkesson states that "[a] potential limitation of anti-IL-8 therapy is that, like any anti-inflammatory therapy, it might inhibit the host immunity and increase the risk of infection" (page 114, right column, full paragraph). In view of this disclosure, the skilled artisan would avoid administering an anti-IL-8 antibody to patients suffering from indirectly caused acute lung injury because doing so would likely compound the systemic causes of the injury. Specifically, the skilled artisan reading Folkesson would not wish to further aggravate a systemic event which indirectly causes acute lung injury, like septicemia, by administering an agent which suppresses the immune system and further degrades systemic function.

Thus, it is clear that the claimed subject matter does not necessarily flow from Folkesson because the document in fact discloses that administration of an anti-IL-8 antibody could aggravate the indirect causes of acute lung injury. Accordingly, it is respectfully requested that the rejection of the claims under 35 U.S.C. §102 be withdrawn. It is also respectfully requested that the rejection of the claims under 35 U.S.C. §103 over Folkesson be withdrawn because Folkesson teaches away from administering an anti-IL-8 antibody.

In the unlikely event that the transmittal letter is separated from this document and the Patent Office determines that an extension and/or other relief is required, applicant petitions for any required relief including extensions of time and authorizes the Assistant Commissioner to charge the cost of such petitions and/or other fees due in connection with the filing of this document to **Deposit Account No. 03-1952** referencing docket no. 350292000500. However, the Commissioner is not authorized to charge the cost of the issue fee to the Deposit Account.

Respectfully submitted,

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Search Results: Taber's Medical Encyclopedia

sepsis

The spread of an infection from its initial site to the bloodstream, initiating a systemic response that adversely affects bloodflow to vital organs. Bacterial infections are the most common source of initial infection, but sepsis also occurs with fungal, parasitic, and mycobacterial infections, particularly in immunocompromised patients. The number of patients with moderate to severe sepsis has increased significantly over the past 20 years.

PATHOLOGY: Organisms that enter the body through skin or the respiratory, genitourinary, or gastrointestinal tracts damage local cells and stimulate both the inflammatory and cell-mediated immune responses, resulting in the release of cytokines, which enhance immune defenses. When the organism overwhelms local defenses and enters the bloodstream, the resulting condition is called *septicemia*. Depending on the organism involved, septicemia may be referred to as bacteremia, fungemia, or viremia.

As organisms circulate, more and more phagocytes and lymphocytes are drawn into the body's defense, and more cytokines, particularly interleukins 1 and 2, tumor necrosis factors, and gamma interferon and other chemical mediators such as leukotrienes, kinin, and complement are released or activated. It is these regulatory proteins, affecting the cardiovascular, central nervous system, bone marrow, and other organ systems, that produce systemic signs and symptoms. Antigenic factors associated with the organisms, such as gram-negative bacterial endotoxin, also stimulate cytokine release.

Pathogens leave the bloodstream or are deposited on blood vessel walls as part of immune complexes, initiating inflammation at other body sites. The extent of local and systemic tissue damage depends on the virulence of the organism, the quality of the host's defenses, and the effectiveness of antimicrobial and supportive therapy. At times, sepsis may inhibit immune responses, particularly when it is caused by viral infections. If host defenses continue to be overwhelmed and blood flow to organ systems is disrupted, septic shock, also known as sepsis syndrome, occurs. SEE: *cytokine*; *disseminated intravascular coagulation*; *infection*; *interferon*; *interleukin*; *tumor necrosis factor*.

SYMPTOMS: Symptoms of sepsis are a white blood cell count greater than 12,000 cells/mm or less than 4000 cells/mm, with increased bands (immature neutrophils); temperature greater than 38°C; rapid respiration; hypotension; and tachycardia. Blood, body fluid, and infection site cultures are often negative. Blood cultures are most likely to grow the causative organism if obtained when the patient's temperature is elevated because fever is the result of interleukin-1 release by macrophages when the organism is in the bloodstream.

TREATMENT: Broad-spectrum antibiotic therapy is used initially. An extended-spectrum penicillin (e.g., piperacillin) or third-generation cephalosporin (e.g., cefotaxime) plus an aminoglycoside (e.g., gentamycin) is administered until the results of blood, urine, sputum, or wound cultures indicate the causative organism and its sensitivity to specific antimicrobial drugs.

puerperal sepsis Any infection of the genital tract occurring during the puerperium or as a complication of abortion. This disease is presumed to be present when the temperature is 38°C (100.4°F) on any two consecutive days, exclusive of the first 24 hr postpartum, if no other causes of fever are apparent. This is a polymicrobial infection caused by a wide variety of bacteria. The establishment of careful techniques of asepsis and hygiene in maternity wards has effectively reduced the importance of this disease as a cause of death in the puerperium. Although this complication of childbirth has been reduced dramatically with an understanding of asepsis and the use of antibiotics to treat infections, the dangers still exist. In home births, a trend particularly popular among many young women, the same aseptic precautions must apply as do in hospitals and birthing centers. Although licensed midwives are educated in aseptic techniques, sometimes attendants engaged by parents to attend home births are not professionally prepared and may not understand the problems and complications that may arise. SYN: *childbed fever*. SEE: *Nursing Diagnoses Appendix*.

DIAGNOSIS: Diagnosis is made on the basis of clinical findings consistent with infection of the genital tract, including fever and pain and tenderness of the lower abdominal area and genital tract.

•SYMPTOMS: The onset may be gradual or sudden. The patient begins to have general malaise, headache, chilly sensations or shaking chills, and a rise in temperature. The uterus is tender, and there is some abdominal distention.

PATHOLOGY: In minor cases of ulceration, the vaginal tract is covered by a dirty membrane. In streptococcal and staphylococcal infections, the endometrium is smooth and the lymphatics are congested with the invading organisms. As a rule, the uterine cavity is filled with very little lochia. The uterus shows poor involution. If the infection extends farther beyond the uterus, the parametrium or cellular tissues show edema, inflammation, and in some cases purulent infiltration. Extension of the process to the veins produces infectious thrombi, which in turn produce localized abscesses in other parts of the body.

TREATMENT: Treatment includes appropriate antibiotic, incision and drainage if abscess forms, and supportive therapy.